Amendments to the Specification:

Please amend the specification as follows:

Please replace the paragraph starting on page 13, line 3, with the following paragraph:

- - 11. The wells were then developed with ~~BluPhos~~ BluPhos™ (Kirkegaard and Perry Laboratories, Gaithersburg, Maryland). Reaction product was allowed to develop in the dark at room temperature, and read at 630 nm within 15-30 minutes. - -

Please replace the paragraph starting on page 20, line 13, with the following paragraph:

- - Previous data established an increase in MIF transcription in aggressive CaP cells (Meyer-Siegler (2000a), *J. Interferon and Cytokine Res.* 20(9): 769-778). At the time a molecular mechanism for the increased MIF mRNA concentrations was not known. A recent publication associated a MIF SNP in the MIF promoter region with systemic juvenile arthritis (Donn et al.(2001), *Arthritis and Rheum.* 44: 1782 – 1785) and the creation of an AP-4 transcription factor-binding site. The presence of an additional transcription factor binding site results in increased MIF gene expression and could therefore be associated with prostate tumor aggressiveness. Analysis of this region in normal, BPH-1, LNCaP, C4-2b, DU-145 and PC-3 cells was undertaken. Genomic DNA was isolated from cells and 0.1 μg of DNA amplified by PCR with the following primers 5'ACTAAGAAAGACCCGAGGC3' (SEQ ID NO: 1) and 5'GGGGCACGTTGGTGTTTAC3' (SEQ ID NO: 2) for 30 cycles of 95°C for 1 min, 60°C for 1 min and 72°C for 1 min. The resulting 366 bp PCR products were purified and 1 μg of PCR product digested with 3U Alu I overnight. Resulting restriction digests were separated by agarose electrophoresis and photographed. This SNP is present in the more aggressive CaP cell lines

(C4-2b, DU-145 and PC-3), but absent from normal and the less aggressive LNCaP cell line. ~~GenBank~~ GenBank™ analysis determined the presence of an expressed sequence tag for AP-4 in the prostate (A1669905). These data suggest that the MIF polymorphism is associated with a positive role in gene expression and provide additional evidence of a functional SNP in the 3' regulator region of the gene. This is a potential genetic mechanism to explain the increased MIF expression seen in aggressive Gleason score tumors (Meyer-Siegler et al. (2002)) and CaP cell lines (Meyer-Siegler (2000a); Meyer-Siegler (2000b); Meyer-Siegler (2001), *Molec. Med.* 7: 850-860). - -

Please replace the paragraph starting on page 22, line 4, with the following paragraph:

- - MALDI-TOF mass spectroscopy and Edman degradation tryptic peptide sequence analysis was used to identify components of this novel protein complex. The complex proteins are N-terminal blocked, precluding less expensive protein sequencing from PVDF membranes. One protein identified is A20, a cytokine-inducible zinc finger protein, which regulates NF-κβ activity (Heyninck and Beyaert (1999), *FEBS Lett.* 442: 147-150). Another is an unknown 70-kDa protein with a peptide sequence GAAKKGAVGGI (SEQ ID NO: 3). ~~SwissPro~~ SwissPro™ database search, revealed no known homologus protein sequences to the unknown protein peptide. A third component of this complex has been identified as human glandular kallikrein-2 (hK2). These new data support those of a previous study, which documented the association of MIF and hK2 in experiments designed to purify hK2 from human seminal fluid (Meinhardt et.al. (1999), *J. Cell Sci.* 112: 1337-1344). Kallikrein gene family proteins are serine proteases (Yousef and Diamandis (2001), *Endocr Rev.* 22: 184-204). In the human prostate the KLK3 kallikrein gene encodes PSA (aka human kallikrein 3, hK3) (Riegman et al. (1992),

Genomics 14:6-11). hK2 has trypsin like activity and can activate the pro-form of PSA to its active form (Kumar et.al. (1997), *Cancer Res.* 57:3111-3114). Expression of KLK2, the gene encoding the hK2 protein, is up-regulated in prostate carcinoma (Darson et.al. (1997), *Urology* 49:857-862). hK2 may participate in prostate growth factor and cytokine networks (Rittenhouse et.al. (1998), *Crit. Rev. Clin. Lab. Sci.* 35:275-368). These data are intriguing given the established interaction between PSA and hK2 and the latter's apparent interaction with cytokines, such as MIF. A20 identity in the MIF complex was confirmed by coimmunoprecipitation pull down experiments. - -